

Technical University of Denmark



Identification of a novel immunoregulatory signaling pathway exploited by M. tuberculosis in dendritic cells

Laursen, Janne Marie; Schoof, Erwin; Søndergaard, Jonas Nørskov; Linding, Rune; Pedersen, Susanne Brix

Publication date:
2013

Document Version
Publisher's PDF, also known as Version of record

[Link back to DTU Orbit](#)

Citation (APA):
Laursen, J. M., Schoof, E., Søndergaard, J. N., Linding, R., & Pedersen, S. B. (2013). Identification of a novel immunoregulatory signaling pathway exploited by M. tuberculosis in dendritic cells. Abstract from 15th International Congress of Immunology, Milan, Italy.

DTU Library

Technical Information Center of Denmark

General rights

Copyright and moral rights for the publications made accessible in the public portal are retained by the authors and/or other copyright owners and it is a condition of accessing publications that users recognise and abide by the legal requirements associated with these rights.

- Users may download and print one copy of any publication from the public portal for the purpose of private study or research.
- You may not further distribute the material or use it for any profit-making activity or commercial gain
- You may freely distribute the URL identifying the publication in the public portal

If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.



 [Print this Page for Your Records](#)

[Close Window](#)

Control/Tracking Number: 2013-LBA-5531-ICI

Activity: Late Breaking Abstract

Current Date/Time: 6/30/2013 6:50:09 AM

Identification of a novel immunoregulatory signaling pathway exploited by M. tuberculosis in dendritic cells

Author Block: Janne Marie Laursen, E. Schoof, J.N. S ndergaard, R. Linding, S. Brix;
Kgs. Lyngby, Denmark

Abstract:

The causative agent of tuberculosis, M. tuberculosis, has infected over a third of the world's population and the persistence of latent infections poses a massive burden to health care systems and human well-being. The dendritic cell (DC) plays a crucial role in shaping the nature of the adaptive immune response after exposure to pathogens, and the interaction between M. tuberculosis and the dendritic cell is of profound importance for the course of infection. During their interaction, the DC is exposed to multiple M. tuberculosis-derived ligands recognized by a range of pattern recognition receptors, but the result is typically an immune response that is not very effective at clearing the bacteria from the host. The reason why the induced immune response is ineffective at clearing the bacteria is not fully understood, but clues may be given in the signaling pathways induced in DCs upon M. tuberculosis-exposure.

High resolution LC-MS/MS was used for a global analysis of the proteome and the phospho-proteome in human DCs upon stimulation with intact M. tuberculosis or purified lipopolysaccharide (LPS). Data were analyzed using MaxQuant and Python, and the algorithm NetworKIN was used for prediction of kinases responsible for the observed phosphorylation sites.

Multiple phosphorylation sites and protein kinases were identified that validate previously identified intracellular signaling structures induced in DCs by M. tuberculosis. Importantly, from the MS data analysis, FMS-related tyrosine kinase 3 (FLT3), that signals through JAK2 and STAT3, was identified as a novel protein kinase potentially activated in DCs by M. tuberculosis.

;

Author Disclosure Information: J. Laursen: None. E. Schoof: None. J.N. S ndergaard: None. R. Linding: None. S. Brix: None.

Keyword (Complete): Intracellular signaling ; Dendritic cells ; Immune regulation ; Systems biology

Topics (Complete): 2.12 Signalling in immune cells ; 4.05 Immunity to mycobacterial infection ; 1.09 Dendritic cell differentiation and function

Submission Fee (Complete):

I reside in an OECD country: Yes

I confirm that the information regarding my residency is accurate and that I have completed the payment if relevant. (required) : True

Status: Complete

ICI 2013 Abstract Submission c/o Vienna Medical Academy

Alserstrasse 4, 1090 Vienna Austria

Phone: +43 1 405 13 83 13

Fax: +43 1 407 82 74

[Leave OASIS Feedback](#)

Powered by [OASIS](#), The Online Abstract Submission and Invitation System SM

  1996 - 2013 [Coe-Truman Technologies, Inc.](#) All rights reserved.

